4 Magnetic Interventions for Gastroesophageal Reflux

Luigi Bonavina

Current therapy for gastroesophageal reflux disease (GERD) is generally reported to be overall unsatisfactory by gastroenterologists, surgeons, and patients. About 40% of patients are resistant or only partial responders to proton-pump inhibitors (PPI) therapy [1, 2], and even doubling the dose may be inadequate to relieve regurgitation and improve quality of life. In addition, there are growing concerns over the long-term consequences of chronic acid suppression (reduced vitamin B12 and magnesium absorption, interaction with clopidogrel, risk of *Clostridium difficile* infection, hypergastrinemia, enterochromaffin-like cell hyperplasia, parietal cell hypertrophy leading to rebound acid hypersecretion, and risk of gastric cancer) [3–5]. Lastly, PPI therapy does not have any direct pharmacologic impact on the dynamics of the lower esophageal sphincter (LES) and the crural diaphragm. Persistent non-acid reflux and nocturnal acid breakthrough can still occur despite maximal PPI therapy and may lead to volume regurgitation with pulmonary aspiration and Barrett's metaplasia, the major risk factor for esophageal adenocarcinoma [6, 7].

Surgical therapy has the potential to cure GERD by reinforcing both the intrinsic (crural diaphragm) and the extrinsic sphincter (LES). Because of equivocal evidence and lack of robust and high-quality randomized trials, current guidelines suggest that the choice of an antireflux procedure should be left to the discretion of the individual surgeon and best suited to the individual patient [8–10]. The laparoscopic Nissen fundoplication remains the current gold standard and has been shown to be safe, effective, and durable when performed in specialized centers [11]. Systematic review and meta-analyses [12] and randomized clinical trials [13] suggest that the Toupet fundoplication provides equivalent results in terms of reflux control and a lower rate of side effects compared to the Nissen fundoplication, especially in patients with defective esophageal body motility and in those with increased esophageal hypersensitivity.

Despite the remarkably low incidence of morbidity and mortality rates, fundoplication is underused due to the perception of long-term side effects and fear of failure [14]. Also, variability in clinical outcomes related to inter-individual surgical expertise and/or unvalidated technical modifications [15] has limited the adoption of this procedure, especially in patients with early-stage GERD. Patients undergoing a Nissen fundoplication are especially at risk for potential side effects of the procedure such as bloating, the inability to belch and vomit, and the occurrence of persistent dysphagia that may occasionally require revisional surgery [16]. These are the main reasons why gastroenterolo-

L. Bonavina (✉)
Division of General and Foregut Surgery, Department of Biomedical Sciences for Health, University of Milano School of Medicine, IRCCS Policlinico San Donato, Milan, Italy
e-mail: luigi.bonavina@unimi.it

M. Gagner (ed.), *Magnetic Surgery*, https://doi.org/10.1007/978-3-030-73947-8_4

gists tend to refer for fundoplication only patients with long-lasting severe disease and large hiatal hernias.

A downward trend in the utilization of surgical fundoplication was noted in the USA over the past decade [17–19]. The decline in surgical volume has been attributed to the perceived risk of fundoplication failure, to the availability of over-the-counter PPI and endoscopic therapies, and to the rise of bariatric surgery. Paradoxically, underutilization of antireflux procedures is in contrast with the increasing recognition of GERD as a progressive disease leading to carditis, cardiac metaplasia, intestinal metaplasia, and eventually adenocarcinoma of the distal esophagus [20, 21]. The limitations of both PPI therapy and fundoplication have led many patients and clinicians either to tolerate a lifetime drug dependence with incomplete symptom relief or to undertake the risk of a surgical procedure that alters gastric anatomy, may have side effects, and may deteriorate over time. The Linx™ Reflux Management System is an FDA-approved device designed to provide a permanent solution to gastroesophageal reflux disease by augmenting the LES barrier with a standardized laparoscopic procedure. The Linx can be used with the intent to prevent progression of early-stage GERD or to treat established and more advanced disease associated to hiatus hernia.

Magnetic Sphincter Augmentation

The Linx is a mechanical device designed to augment the physiologic barrier to reflux by magnetic force. The device is manufactured in different sizes and consists of a series of biocompatible titanium beads with magnetic cores hermetically sealed inside. The beads are interlinked with independent titanium wires to form a flexible and expandable ring with a Roman arch configuration (Fig. 4.1). At rest, each bead is in contact with adjacent beads. The beads can move independently of the adjacent beads, creating a dynamic implant that does not compress the esophagus and does not limit its range of motion upon swallowing, belching, and vomiting (Fig. 4.2). Rather, the Linx device prevents reflux by limiting distension of the esophagogastric junction in response to challenges of intragastric pressure. Separation of the beads occurs when intragastric pressure overcomes the magnetic attraction force and is independent of the number of beads contained in the device. The Linx, while augmenting the LES, allows for expansion to accommodate a swallowed bolus or the escape of elevated gastric pressure associated with belching or vomiting. During the healing process after implantation, the device is encapsulated in fibrous tissue but is not incorporated in the esophageal wall [22]; this makes possible to remove the device without damaging the esophagus. The

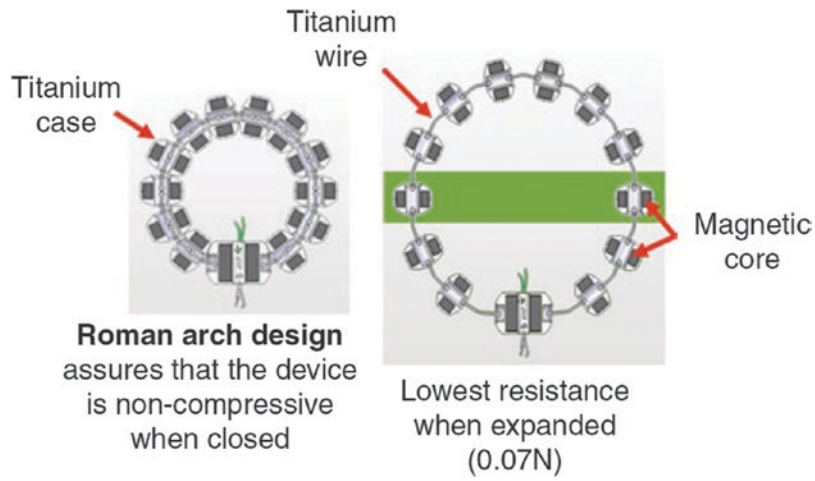

Fig. 4.1 Design of the Linx device. (From Mihura and Louie [48]. Reprinted with permission)

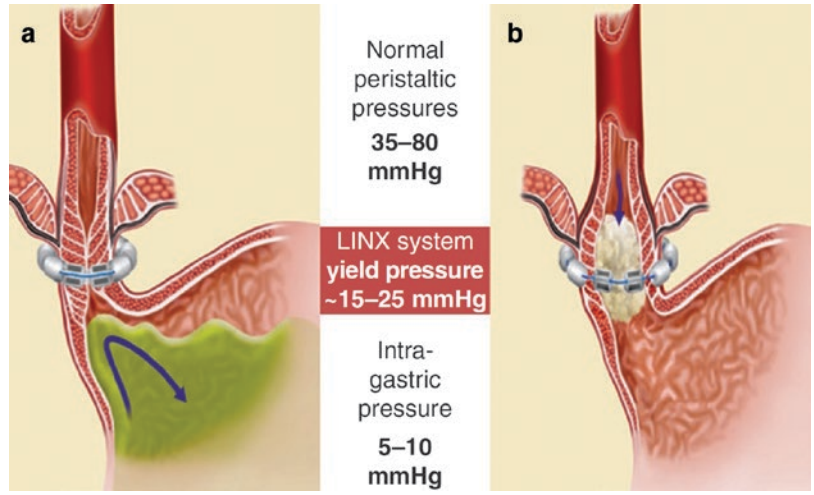

Fig. 4.2 Mechanism of action of the Linx device in the closed (**a**) and in the open (**b**) position. (From Mihura and Louie [48]. Reprinted with permission)

Linx has recently received magnetic resonance imaging (MRI) approval for scanning in systems up 1.5 Tesla.

Preoperative Work-Up

The preoperative assessment of patients who are candidates for a Linx procedure is essentially similar to any other antireflux intervention. Routine testing includes a barium swallow study, upper gastrointestinal endoscopy with biopsies, esophageal manometry, and esophageal pH monitoring. In selected patients, gastric emptying scintigraphy may be performed.

Surgical Technique

Compared to the current surgical standard, the Linx procedure in patients without hiatus hernia requires minimal dissection and preservation of the phrenoesophageal ligament [23]. The device is implanted with a standard laparoscopic approach under general anesthesia. There is no available data supporting the use of single-port access, three-dimensional camera, or robotics for performance of the Linx procedure. The steps of the procedure are illustrated in Fig. 4.3. Surgical dissection begins by dividing the peritoneum on the anterior surface of the gastroesophageal junction below the insertion of the inferior leaf of the phrenoesophageal ligament and above the junction of the hepatic branch to the anterior vagus nerve. The lateral surface of the left crus is dissected from the posterior fundic wall without dividing the short gastric vessels. The gastro-hepatic ligament is opened above and below the hepatic branch of the anterior vagus nerve to facilitate preparation of the retro-esophageal window. Gentle dissection from the right side is made toward the left crus just above the crural decussation to identify the posterior vagus nerve. A tunnel is created between the vagus and the posterior esophageal wall, and the esophagus is encircled with a Penrose drain. The circumference of the esophagus is measured to determine the proper size of the Linx device to be implanted. The sizing tool is a laparoscopic instrument with a soft, circular curved tip actuated by coaxial tubes through a handset. The handset contains a numerical indicator that corresponds to the size range of the Linx device. The sizing tool is placed around the esophagus in the tunnel dissected between the esophageal wall and the posterior vagus nerve bundle. The Linx device of appropriate size is introduced through the tunnel, and the opposing ends are brought to the anterior surface of the esophagus and simply connected together by engaging the two clasps. The decision to proceed with a posterior hiatal repair depends on the severity of GERD as assessed preoperatively, and the size of the hernia that is confirmed intraoperatively. Occasionally, simple correction of crura diastasis with one to two nonabsorbable stitches may be indicated. However, in the presence of hiatal hernia greater than 2 cm, division of

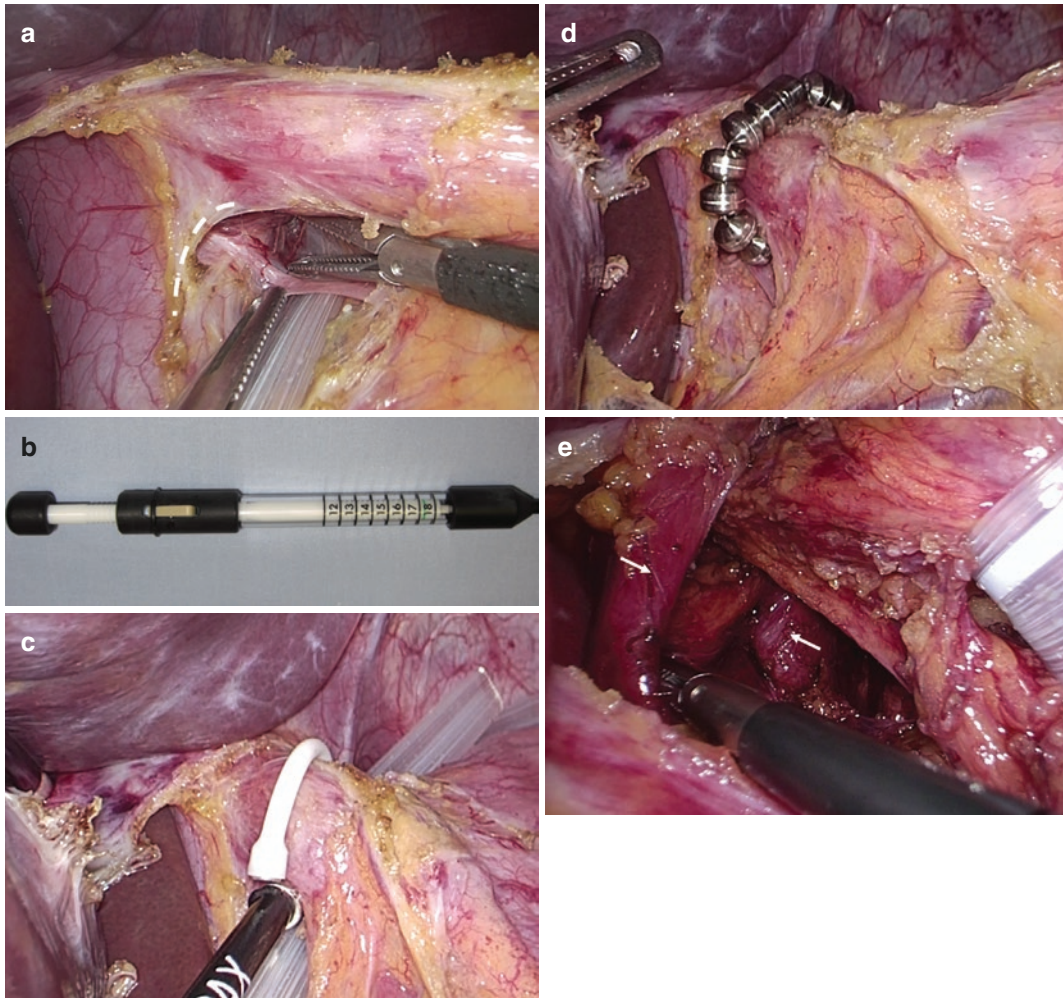

Fig. 4.3 Surgical steps of the Linx procedure. (**a**) The phrenoesophageal ligament is preserved and a tunnel is created between the posterior vagus nerve and the esophageal wall. (**b**) The circumference of the esophagus is measured using a special sizing tool. (**c**) Linx device locked in front of the esophagus after engagement of the two clasps. (**d**) No hiatus closure (minimal dissection). (**e**) Formal mediastinal dissection and posterior crura repair

the phrenoesophageal ligament and full mediastinal dissection is recommended to obtain an adequate length of intra-abdominal esophagus.

Postoperative Management

Patients are discharged the same day of surgery or on the first postoperative day after obtaining a chest film to control the position of the Linx (Fig. 4.4). Patients are encouraged to chew well, eat five small-volume meals during the day, and gradually discontinue PPI therapy. Dysphagia is considered normal during the first 3 months after surgery, with a peak generally occurring between the third and the sixth postoperative week. In such circumstances, a temporary switch to a semiliquid diet is recommended. Persistent dysphagia may occasionally require a short course of steroids and/or endoscopic pneumatic dilation.

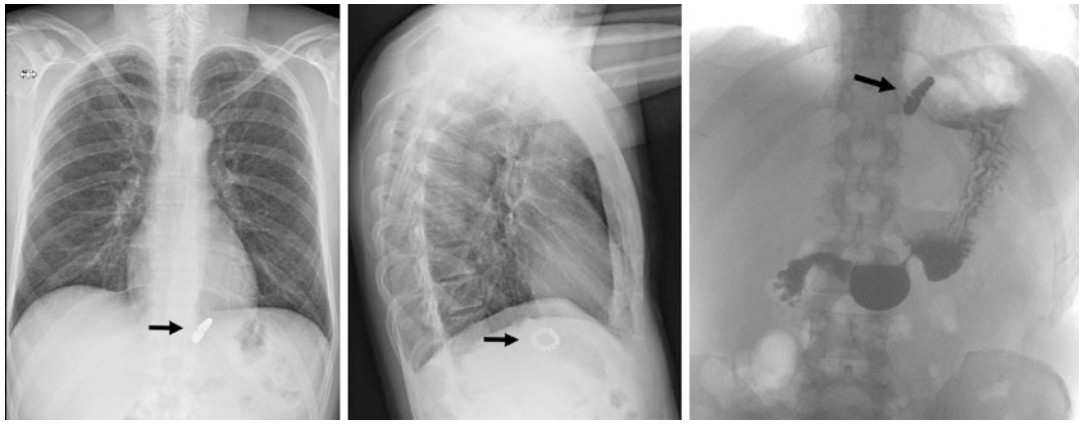

Fig. 4.4 Chest film and barium swallow study after Linx implant

Overview of Clinical Experience

Since the first human implantation in 2007, all reported studies investigating the long-term clinical outcomes of the Linx device have confirmed a high rate of symptom relief, discontinuation of PPI therapy, objective reduction of esophageal acid exposure, and improved quality of life. The feasibility study included 44 patients implanted with the Linx at 4 study centers in the USA and in Europe between February 2007 and October 2008; the short-term, midterm, 4-year, and final results of this study have been previously published [23–26]. Patients served as their own control to assess the effect of treatment on symptoms, use of PPI, and esophageal acid exposure. The primary criteria for inclusion in the feasibility trial were age >18 and <85 years, typical reflux symptoms at least partially responsive to PPI therapy, abnormal esophageal acid exposure, and normal contractile amplitude and wave form in the esophageal body. The primary criteria for exclusion from the trial were history of dysphagia, previous upper abdominal surgery, previous endoluminal antireflux procedures, sliding hiatal hernia >3 cm, esophagitis >grade A, and/or the presence of histologically documented Barrett's esophagus. Patients with abnormal manometric findings (distal esophageal contraction amplitude of less than 35 mmHg on wet swallows or <70% propulsive peristaltic sequences) were also excluded. All Linx devices were successfully implanted via a standard laparoscopic approach. The median operative time was 40 minutes. No intraoperative complications occurred. Patients were instructed to resume a regular diet after a chest film and radiological assessment of the esophageal transit were performed. Forty-three percent of patients complained of mild dysphagia during the postoperative period; in all individuals the symptom resolved by 90 days without treatment. Thirty-three patients (75%) were followed up to 5 years. The mean total GERD-HRQL score off PPI significantly decreased from 25.7 at baseline to 2.9, and 94% of patients had a greater than 50% reduction in the total score compared to baseline. Complete cessation of PPI or a reduction of 50% or more of the daily dose was achieved by 88% and 94% of patients, respectively, and 91% of patients declared to be satisfied with their current condition. Esophageal pH testing was completed in 20 patients at 5 years: 85% of patients either achieved normal esophageal acid exposure or had at least a 50% reduction from baseline, and 70% of patients achieved normalization of the pH profile. Three patients were explanted: one because of persistent dysphagia, one because of the need to undergo magnetic resonance imaging, and the last one elected to have a Nissen fundoplication for persisting GERD symptoms. All removals were safely performed via laparoscopy.

Similar rigorous inclusion criteria and perioperative subjective and objective assessment

were used for a larger multi-institutional study involving 100 patients at 13 centers [27]. Significant improvements were seen in GERD-related quality of life, regurgitation, and esophageal acid exposure. Use of PPI dropped to 13% at 3 years and patient satisfaction with reflux control increased to 94% after implantation. Importantly, these positive results were stable showing no degradation over the study time period. Although 14% of patients reported bloating after implantation, no patients rated this symptom as severe. Patients retained their ability to belch and vomit. Dysphagia was present to some extent in 68% of patients but decreased to 4% by 3 years. Five percent of patients rated the dysphagia as severe, and the device was removed in three of them with complete symptom resolution.

Two single-center studies have further validated the efficacy of the Linx procedure. In Milan, Italy, 100 consecutive patients underwent Linx implantation between 2007 and 2012. The median implant duration was 3 years. There was a significant reduction of acid exposure time and improvement of GERD-HRQL score; freedom from daily dependence on PPI was achieved in 85% of the patients [28]. Another study from the USA, including 66 patients with an average follow-up of 5.8 months, showed similar satisfactory results [29].

Three recent case-control studies found comparable control of reflux symptoms after surgical fundoplication or Linx implant. However, in the Nissen fundoplication group, there was a higher rate of patients with inability to belch and vomit, along with more severe gas-bloat symptoms, whereas quality of life scores were similar in patients treated either by Linx or Toupet fundoplication [30–32]. A recent meta-analysis comparing Linx and fundoplication reported that the former was associated with less gas-bloat symptoms and an increased ability to vomit and belch, while PPI suspension rate, dysphagia requiring endoscopic dilatation, and GERD-HRQL were similar in the two patient groups [33].

It has been reported that the short-term results of the Linx procedure combined with systematic crural repair appear more favorable compared to Linx alone regardless of the size of hiatus hernia [34–38]. A multivariate logistic regression analysis confirmed that full mediastinal dissection with restoration of intra-abdominal esophageal length and crural repair was most likely to normalize esophageal acid exposure [39].

Regression of Barrett's has been observed in 72% of patients at 1 year after Linx implant; interestingly, patients with short-segment intestinal metaplasia in whom esophageal acid exposure reversed to normal were more likely to achieve regression [40]. It appears that early recognition of GERD is critical to prevent long-term complications, even in patients under continuous acid-suppressive medication [41]. A retrospective single-center review of 553 patients showed that the factors associated with a favorable outcome of the Linx procedure are age younger than 45 years, male sex, GERD-HRQL>15, and an abnormal DeMeester score [42].

Safety Profile

Concerns regarding the safety of this operation, especially the fear of erosions, stem from past adverse experience with the Angelchik device and, more recently, with the gastric banding device. An analysis of the safety profile of the first 1000 worldwide implants in 82 hospitals showed 1.3% hospital readmission rate, 5.6% need of postoperative endoscopic dilations, and 3.4% reoperation rate [43]. All reoperations were performed electively for device removal. The most commons symptoms were dysphagia and recurrence of reflux symptoms. In addition, 7% of patients enrolled in the US multicenter single-arm trial had the device removed due to persistent dysphagia in four, vomiting in one, chest pain in one, and reflux in one [44]. A study reported the results of reoperations for laparoscopic Linx removal in a series of 164 consecutive patients [45]. The reoperation rate was 6.7%, and a partial fundoplication was most commonly associated to Linx removal. The main presenting symptoms requiring device removal was recurrence of heartburn or regurgitation in 46%, dysphagia in 37%, and chest pain in 18%. In two patients

(1.2%), full-thickness erosion of the esophageal wall with partial endoluminal penetration of the device occurred (Fig. 4.5). The median implant duration was 20 months, with 82% of the patients being explanted between 12 and 24 months after the index operation. Operative time ranged from 25 to 150 minutes and postoperative course was uneventful. At the latest follow-up (12–58 months), the GERD-HRQL score was normalized in all patients.

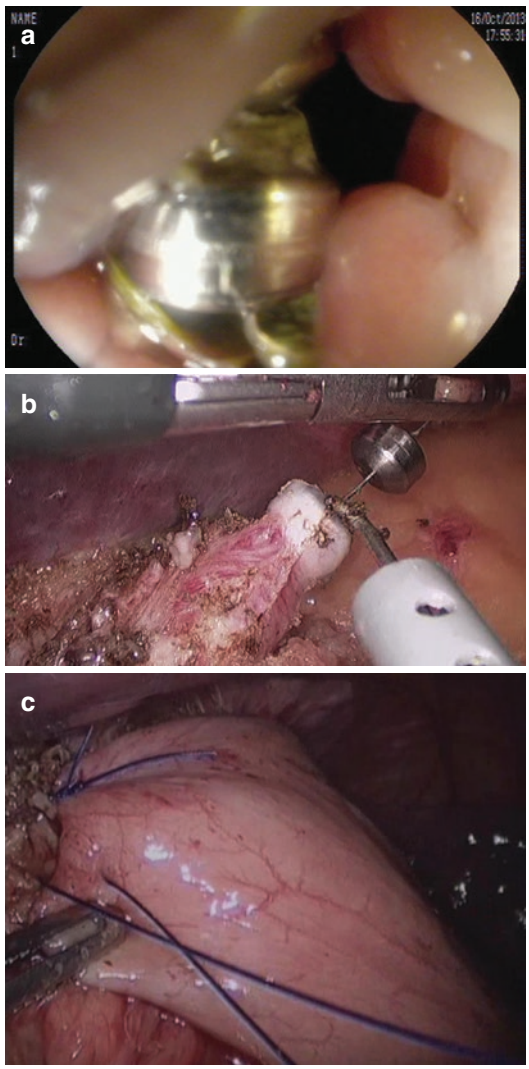

Fig. 4.5 Endoscopic view of erosion of Linx device (**a**) requiring laparoscopic removal (**b**) and Dor fundoplication (**c**)

Conclusion

The Linx procedure was developed to address the unmet needs of patients with unsatisfactory response to medical therapy and those with early-stage GERD who would not usually be considered ideal candidates for fundoplication [23, 46–49]. The Linx is highly effective in reducing typical symptoms with a favorable side-effect profile and therefore provides a standardized and physiological alternative to fundoplication. A randomized clinical trial has shown the superiority of Linx compared to daily PPI therapy in controlling moderate to severe regurgitation and reducing esophageal acid exposure [50]. Safety issues such as device erosions or migrations have been rare and not associated with mortality. The Linx can be easily removed if necessary, thereby preserving the option of fundoplication in the future. Among the potential limitations of this procedure are the current contraindication to undergo scanning in MRI systems >1.5 Tesla and the potential long-term consequences of a permanent foreign body implant. Randomized trials are needed to definitively assess the effectiveness of the procedure and to establish at which stage of disease severity magnetic sphincter augmentation may prove superior to fundoplication.

References

1. Toghanian S, Johnson DA, Stalhammar NO, Zerbib F. Burden of gastro-oesophageal reflux disease in patients with persistent and intense symptoms despite proton pump inhibitor therapy: a post-hoc analysis of the 2007 national health and wellness survey. Clin Drug Investig. 2011;31:703–15.
2. Kahrilas PJ, Howden CW, Hughes N. Response of regurgitation to proton pump inhibitor therapy in clinical trials of gastroesophageal reflux disease. Am J Gastroenterol. 2011;106:1419–25.
3. Heidelbaugh JJ, Kim AH, Chang R, et al. Overutilization of proton-pump inhibitors: what the clinician needs to know. Ther Adv Gastroenterol. 2012;5(4):219–32.
4. McColl K, Gillen D. Evidence that proton-pump inhibitor therapy induces the symptoms it is used to treat. Gastroenterology. 2009;137:20–2.
5. Poulsen AH, Christensen S, McLaughlin JK, et al. Proton pump inhibitors and risk of gastric cancer:

a population-based cohort study. Br J Cancer. 2009;100:1503–7.
6. Lord R, DeMeester S, Peters J, et al. Hiatal hernia, lower esophageal sphincter incompetence, and effectiveness of Nissen fundoplication in the spectrum of gastroesophageal reflux disease. J Gastrointest Surg. 2009;13:602–10.
7. Malfertheiner P, Nocon M, Vieth M, et al. Evolution of gastro-oesophageal reflux disease over 5 years under routine medical care – the ProGERD study. Aliment Pharmacol Ther. 2012;35:154–64.
8. Stefanidis D, Hope WW, Kohn GP, et al. Guidelines for surgical treatment of gastroesophageal reflux disease. Surg Endosc. 2010;24:2647–69.
9. Kohn GP, Price RR, DeMeester SR, et al. Guidelines for the management of hiatal hernia. Surg Endosc. 2013;27:4409–28.
10. Fuchs KH, Babic B, Breithaupt W, et al. EAES recommendations for the management of gastroesophageal reflux disease. Surg Endosc. 2014;28:1753–73.
11. Galmiche JP, Hatlebakk J, Attwood S, et al. Laparoscopic antireflux surgery vs esomeprazole treatment for chronic GERD: the LOTUS randomized clinical trial. JAMA. 2011;305(19):1969–77.
12. Broeders JA, Mauritz FA, Ahmed Ali U, et al. Systematic review and meta-analysis of laparoscopic Nissen (posterior total) versus Toupet (posterior partial) fundoplication for gastro-oesophageal reflux disease. Br J Surg. 2010;97:1318–30.
13. Hakanson BS, Lundell L, Bylund A, Thorell A. Comparison of laparoscopic 270° posterior partial fundoplication vs total fundoplication for the treatment of gastroesophageal reflux disease. A randomized clinical trial. JAMA Surg. https://doi.org/10.1001/jamasurg.2019.0047.
14. Niebisch S, Fleming FJ, Galey KM, et al. Perioperative risk of laparoscopic fundoplication: safer than previously reported – analysis of the American College of Surgeons National Surgical Quality Improvement Program 2005 to 2009. J Am Coll Surg. 2012;215:61–9.
15. Richter JE, Dempsey DT. Laparoscopic antireflux surgery: key to success in the community setting. Am J Gastroenterol. 2008;103:289–91.
16. Khajanchee YS, O'Rourke R, Cassera MA, et al. Laparoscopic reintervention for failed antireflux surgery: subjective and objective outcomes in 176 consecutive patients. Arch Surg. 2007;142:785–91.
17. Finks JF, Wei Y, Birkmeyer JD. The rise and fall of antireflux surgery in the United States. Surg Endosc. 2006;20:1698–701.
18. Colavita PD, Belyansky I, Walters AL, et al. Nationwide inpatient sample: have antireflux procedures undergone regionalization? J Gastrointest Surg. 2013;17:6–13.
19. Khan F, Maradey-Romero C, Ganocy S, Frazier R, Fass R. Utilisation of surgical fundoplication for patients with gastro-oesophageal reflux disease in the USA has declined rapidly between 2009 and 2013. Aliment Pharmacol Ther. 2016;43:1124–31.
20. Labenz J, Chandrasoma PJ, Knapp LJ, DeMeester TR. Proposed approach to the challenging management of progressive gastroesophageal reflux disease. World J Gastrointest Endosc. 2018;10:175–83.
21. Pinto D, Plieschnegger W, Schneider NI, et al. Carditis: a relevant marker of gastroesophageal reflux disease. Data from a prospective central European multicenter study on histological and endoscopic diagnosis of esophagitis (histoGERD trial). Dis Esophagus. 2019;32(1). https://doi.org/10.1093/dote/doy073.
22. Ganz R, Gostout C, Grudem J, et al. Use of a magnetic sphincter for the treatment of GERD: a feasibility study. Gastrointest Endosc. 2008;67:287–94.
23. Bonavina L, Saino G, Bona D, et al. Magnetic augmentation of the lower esophageal sphincter: results of a feasibility clinical trial. J Gastrointest Surg. 2008;12:2133–40.
24. Bonavina L, DeMeester TR, Fockens P, et al. Laparoscopic sphincter augmentation device eliminates reflux symptoms and normalizes esophageal acid exposure. Ann Surg. 2010;252:857–62.
25. Lipham JC, DeMeester TR, Ganz RA, et al. The Linx reflux management system: confirmed safety and efficacy now at 4 years. Surg Endosc. 2012;26:2944–9.
26. Saino G, Bonavina L, Lipham J, Dunn D, Ganz RA. Magnetic sphincter augmentation for gastroesophageal reflux at 5 years: final results of a pilot study show long-term acid reduction and symptom improvement. J Laparoendosc Adv Surg Tech A. 2015;25:787–92.
27. Ganz RA, Peters JH, Horgan S, et al. Esophageal sphincter device for gastroesophageal reflux disease. N Engl J Med. 2013;368:719–27.
28. Bonavina L, Saino G, Bona D, et al. One hundred consecutive patients treated with magnetic sphincter augmentation for gastroesophageal reflux disease: 6 years of clinical experience from a single center. J Am Coll Surg. 2013;217:577–85.
29. Smith CD, Devault KR, Buchanan M. Introduction of mechanical sphincter augmentation for gastroesophageal reflux disease into practice: early clinical outcomes and keys to successful adoption. J Am Coll Surg. 2014;218:776–81.
30. Louie BE, Farivar AS, Schultz D, et al. Short-term outcomes using magnetic sphincter augmentation versus Nissen fundoplication for medically resistant gastroesophageal reflux disease. Ann Thorac Surg. 2014;98:498–504.
31. Riegler M, Schoppman SF, Bonavina L, et al. Magnetic sphincter augmentation and fundoplication for GERD in clinical practice: one-year results of a multicenter, prospective observational study. Surg Endosc. 2015;29:1123–9.
32. Reynolds J, Zehetner J, Wu P, et al. Laparoscopic magnetic sphincter augmentation vs laparoscopic Nissen fundoplication; a matched-pair analysis of 100 patients. Ann Surg. 2015;221:123–8.
33. Aiolfi A, Asti E, Bernardi D, et al. Early results of magnetic sphincter augmentation versus

fundoplication for gastroesophageal reflux disease: systematic review and meta-analysis. Int J Surg. 2018;52:82–8.
34. Rona KA, Reynolds J, Schwameis K, et al. Efficacy of magnetic sphincter augmentation in patients with large hiatal hernias. Surg Endosc. 2017;31(5):2096–102.
35. Kuckelman JP, Phillips CJ, Hardin MO, et al. Standard vs expanded indications for esophageal magnetic sphincter augmentation for reflux disease. JAMA Surg. 2017;152(9):890–1.
36. Buckley FP, Bell RCW, Freeman K, et al. Favorable results from a prospective evaluation of 200 patients with large hiatal hernias undergoing LINX magnetic sphincter augmentation. Surg Endosc. 2018;32:1762–8.
37. Schwameis K, Nikolic M, Morales Castellano DG, et al. Crural closure improves outcomes of magnetic sphincter augmentation in GERD patients with hiatal hernia. Sci Rep. 2018;8:7319.
38. Tatum JM, Alicuben E, Bildzukewicz N, et al. Minimal versus obligatory dissection of the diaphragmatic hiatus during magnetic sphincter augmentation surgery. Surg Endosc. 2019;33:782–8.
39. Irribarra MM, Blitz S, Wilshire CL, et al. Does treatment of the hiatus influence the outcomes of magnetic sphincter augmentation for chronic GERD? J Gastrointest Surg. 2019;23:1104–12.
40. Alicuben ET, Tatum JM, Bildzukewicz N, et al. Regression of intestinal metaplasia following magnetic sphincter augmentation device placement. Surg Endosc. 2019;33:576–9.
41. Bonavina L, Fisichella PM, Gavini S, Lee YY, Tatum RP. Clinical course of gastroesophageal reflux disease and impact of treatment in symptomatic young patients. Ann N Y Acad Sci. 2020. https://doi.org/10.1111/nyas.14350.
42. Ayazi S, Zheng P, Zaidi AH, et al. Clinical outcomes and predictors of favorable results after laparoscopic magnetic sphincter augmentation: single-institution experience with more than 500 patients. J Am Coll Surg. 2020. doi.org/10.1016. ISSN 1072-7515-20.
43. Lipham JC, Taiganides PA, Louie BE, et al. Safety analysis of first 1000 patients treated with magnetic sphincter augmentation for gastroesophageal reflux disease. Dis Esophagus. 2015;28:305–11.
44. Ganz RA, Edmundowicz SA, Taiganides PA, et al. Long-term outcomes of patients receiving a magnetic sphincter augmentation device for gastroesophageal reflux. Clin Gastroenterol Hepatol. 2016;14:671–7.
45. Asti E, Siboni S, Lazzari V, et al. Removal of the magnetic sphincter device. Surgical technique and results of a single-center cohort study. Ann Surg. 2017;265:941–5.
46. Bonavina L, DeMeester TR, Ganz R. LINX TM reflux management system: magnetic sphincter augmentation in the treatment of gastroesophageal reflux disease. Expert Rev Gastroenterol Hepatol. 2012;6(6):667–74.
47. Worrel SG, Greene CL, DeMeester TR. The state of surgical treatment of gastroesophageal reflux disease after five decades. J Am Coll Surg. 2014;219:819–30.
48. Mihura M, Louie BE. Laparoscopic antireflux surgery: magnetic GEJ augmentation. In: Grams J, Perry K, Tavakkoli A, editors. The SAGES manual of foregut surgery. Cham: Springer; 2019. p. 171–92.
49. Bonavina L, Boyle N, Schoppmann S. The role of magnetic sphincter augmentation in the treatment of gastroesophageal reflux disease. Curr Opin Gastroenterol. 2021. https://doi.org/10.1097/mog.0000000000000748.
50. Bell R, Lipham J, Louie BE, et al. Magnetic sphincter augmentation superior to proton pump inhibitors for regurgitation in a 1-year randomized trial. Clin Gastroenterol Hepatol. 2020;18(8):1736–1743.e2.